```
 ********** STN Columbus **********
**

FILE 'HOME' ENTERED AT 13:32:54 ON 22 SEP 2000

=> file medline embase biosis inpadoc caplus

COST IN U.S. DOLLARS                SINCE FILE    TOTAL
                                    ENTRY SESSION
FULL ESTIMATED COST                   0.30      0.30

FILE 'MEDLINE' ENTERED AT 13:33:44 ON 22 SEP 2000

FILE 'EMBASE' ENTERED AT 13:33:44 ON 22 SEP 2000
COPYRIGHT (C) 2000 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 13:33:44 ON 22 SEP 2000
COPYRIGHT (C) 2000 BIOSIS(R)

FILE 'INPADOC' ENTERED AT 13:33:44 ON 22 SEP 2000
COPYRIGHT (C) 2000 European Patent Office, Vienna (EPO)

FILE 'CAPLUS' ENTERED AT 13:33:44 ON 22 SEP 2000
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2000 AMERICAN CHEMICAL SOCIETY (ACS)

=> e changeux jean-pierre/au

E1      1   CHANGEUX JEAN PIERE/AU
E2    407   CHANGEUX JEAN PIERRE/AU
E3      0 --> CHANGEUX JEAN-PIERRE/AU
E4      1   CHANGEUX M J/AU
E5      2   CHANGEUX P/AU
E6      8   CHANGEUX T/AU
E7      1   CHANGEUX THOAMS/AU
E8      4   CHANGEY NICOLAS/AU
E9      5   CHANGEZ M/AU
E10     1   CHANGEZ MOHAMMED/AU
E11     1   CHANGEZ MOHD/AU
E12     1   CHANGEZI ALTAMASH/AU

=> s e1-e2

L1      408 ("CHANGEUX JEAN PIERE"/AU OR "CHANGEUX JEAN PIERRE"/AU)

=> e picciotto marina/au

E1     94   PICCIOTTO M R/AU
E2      1   PICCIOTTO MARIAN R/AU
E3     13 --> PICCIOTTO MARINA/AU
E4     57   PICCIOTTO MARINA R/AU
E5      1   PICCIOTTO MARINA RACHEL/AU
E6      1   PICCIOTTO R/AU
E7      3   PICCIOTTO ROBERT/AU
E8      2   PICCIOTTO S/AU
E9      2   PICCIOTTO SONDRA/AU
E10     1   PICCIOTTO TED/AU
E11     3   PICCIOTTOC L/AU
E12     1   PICCIOTTOR G/AU

=> s e2-e5

L2      72 ("PICCIOTTO MARIAN R"/AU OR "PICCIOTTO MARINA"/AU OR "PICCIOTTO
        MARINA R"/AU OR "PICCIOTTO MARINA RACHEL"/AU)

=> e bassis alain/au

E1      1   BASSIRLRAD H/AU
E2      1   BASSIROU N/AU
E3      0 --> BASSIS ALAIN/AU
E4      1   BASSIS EFIM/AU
E5      2   BASSIS L M/AU
E6     13   BASSIS M L/AU
E7      1   BASSISSI E/AU
E8      2   BASSISSI FABIO/AU
E9     24   BASSISSI P/AU
E10     1   BASSISSI PAOLA/AU
E11     9   BASSIST L/AU
E12     2   BASSIST L F/AU

=> e bessis alain/au

E1     52   BESSIS A/AU
E2      8   BESSIS A S/AU
E3     21 --> BESSIS ALAIN/AU
E4      1   BESSIS ANDRE/AU
E5      5   BESSIS ANNE SOPHIE/AU
E6      1   BESSIS C/AU
E7     89   BESSIS D/AU
E8      6   BESSIS DANIEL/AU
E9      3   BESSIS DIDIER/AU
E10     1   BESSIS E/AU
E11    11   BESSIS F/AU
E12    21   BESSIS G/AU

=> s e1-e3

L3      81 ("BESSIS A"/AU OR "BESSIS A S"/AU OR "BESSIS ALAIN"/AU)

=> s l1 or l2 or l3

L4      514 L1 OR L2 OR L3

=> s l4 and nicotinic acetylcholine receptor/ab,bi

'AB' IS NOT A VALID FIELD CODE

L5      136 L4 AND NICOTINIC ACETYLCHOLINE RECEPTOR/AB,BI

=> s l5 and promoter/ab,bi

'AB' IS NOT A VALID FIELD CODE
L6      27 L5 AND PROMOTER/AB,BI

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7      11 DUP REM L6 (16 DUPLICATES REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 11 ANSWERS - CONTINUE? Y/(N):y


L7  ANSWER 1 OF 11  BIOSIS  COPYRIGHT 2000 BIOSIS
DUPLICATE 1
AN  1999:222992  BIOSIS
DN  PREV199900222992
TI  Induction of utrophin gene expression by heregulin in skeletal
muscle
    cells: Role of the N-box motif and GA binding protein.
AU  Gramolini, Anthony O.; Angus, Lindsay M.; Schaeffer, Laurent;
Burton,
    Edward A.; Tinsley, Jonathon M.; Davies, Kay E.;
***Changeux,***
  ***  Jean-Pierre***  ; Jasmin, Bernard J. (1)
CS  (1) Department of Cellular and Molecular Medicine, Faculty of
Medicine,
    University of Ottawa, 451 Smyth Road, Ottawa, ON, K1H 8M5
Canada
SO  Proceedings of the National Academy of Sciences of the United
States of
    America, (March 16, 1999) Vol. 96, No. 6, pp. 3223-3227.
    ISSN: 0027-8424.
DT  Article
LA  English
SL  English
AB  The modulation of utrophin gene expression in muscle by the
nerve-derived
    factor agrin plausibly involves the trophic factor ARIA/heregulin.
Here we
    show that heregulin treatment of mouse and human cultured
myotubes caused
    a apprxeq2.5-fold increase in utrophin mRNA levels. Transient
transfection
    experiments with utrophin  ***promoter***  -reporter gene
constructs
    showed that this increase resulted from an enhanced transcription of
the
    utrophin gene. In the case of the  ***nicotinic***
     ***acetylcholine***  ***receptor***  delta and epsilon
subunit genes,
```

heregulin was previously reported to stimulate transcription via a conserved ***promoter*** element, the N-box, which binds the multimeric Ets-related transcription factor GA binding protein (GABP). Accordingly, site-directed mutagenesis of a single N-box motif in the utrophin gene ***promoter*** abolished the transcriptional response to heregulin. In addition, over-expression of heregulin, or of the two GABP subunits in cultured myotubes, caused an N-box-dependent increase of the utrophin ***promoter*** activity. In vivo, direct gene transfer into muscle confirmed that heregulin regulates utrophin gene expression. Finally, electrophoretic mobility shift assays and supershift experiments performed with muscle extracts revealed that the N-box of the utrophin ***promoter*** binds GABP. These findings suggest that the subsynaptic activation of transcription by heregulin via the N-box motif and GABP are conserved among genes expressed at the neuromuscular junction. Because utrophin can functionally compensate for the lack of dystrophin, the elucidation of the molecular mechanisms regulating utrophin gene transcription may ultimately lead to therapies based on utrophin expression throughout the muscle fibers of Duchenne muscular dystrophy patients.

L7 ANSWER 2 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
AN 1998:307358 BIOSIS
DN PREV199800307358
TI Nonmyogenic factors bind ***nicotinic*** ***acetylcholine*** ***receptor*** ***promoter*** elements required for response to denervation.
AU Bessereau, Jean-Louis; Laudenbach, Vincent; Le Poupon, Chantal; ***Changeux, Jean-Pierre (1)***
CS (1) Neurobiol. Mol., UA CNRS D1284, Dep. Biotechnol., Inst. Pasteur 25/28 rue du Dr. Roux, 75724 Paris Cedex 15 France
SO Journal of Biological Chemistry, (May 22, 1998) Vol. 273, No. 21, pp. 12786-12793. ISSN: 0021-9258.
DT Article
LA English
AB Nicotinic acetylcholine receptors (AChRs) belong to a class of muscle proteins whose expression is regulated by muscle electrical activity. In innervated muscle fiber, AChR genes are transcriptionally repressed outside of the synapse, while after denervation they become reexpressed throughout the fiber. The myogenic determination factors (MDFs) of the MyoD family have been shown to play a central role in this innervation-dependent regulation. In the chicken AChR alpha-subunit gene ***promoter*** , two E-boxes that bind MDFs are necessary to achieve the enhancement of transcription following muscle denervation. However, the deletion of ***promoter*** sequences located upstream to these E-boxes greatly impairs the response to denervation (Bessereau, J. L., Stratford-Perricaudet, L. D., Piette, J., Le Poupon, C. and Changeux, J. P. (1994) Proc. Natl. Acad. Sci. U.S.A. 91, 1304-1308). Here we identified two additional cis-regulatory elements of the alpha-subunit gene ***promoter*** that cooperate with the E-boxes in the denervation response. One region binds the Sp1 and Sp3 zinc finger transcription factors. The second region binds at least three distinct factors, among which we identified an upstream stimulatory factor, a b-ZIP-HLH transcription factor. We propose that among MDF-responsive muscle promoters, a specific combination between myogenic and nonmyogenic factors specify innervation-dependent versus innervation-independent promoters.

L7 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1998:318654 BIOSIS
DN PREV199800318654
TI Implication of a multisubunit Ets-related transcription factor in synaptic expression of the ***nicotinic*** ***acetylcholine*** ***receptor*** .
AU Schaeffer, Laurent; Duclert, Nathalie; Huchet-Dymanus, Monique; ***Changeux, Jean-Pierre (1)***
CS (1) CNRS UA D1284 'Neurobiologie Moleculaire', Inst. Pasteur, 25 rue du Dr Roux, F-75724 Paris Cedex 15 France
SO EMBO (European Molecular Biology Organization) Journal, (June 1, 1998) Vol. 17, No. 11, pp. 3078-3090. ISSN: 0261-4189.
DT Article
LA English
AB In adult muscle, transcription of the ***nicotinic*** ***acetylcholine*** ***receptor*** (AChR) is restricted to the nuclei located at the neuromuscular junction. The N-box, a new ***promoter*** element, was identified recently and shown to contribute to this compartmentalized synaptic expression of the AChR delta- and epsilon-subunits. We demonstrate that the N-box mediates transcriptional activation in cultured myotubes and identify the transcription factor that binds to the N-box as a heterooligomer in myotubes and adult muscle. The GABP (GA-binding protein) alpha-subunit belongs to the Ets family of transcription factors, whereas the beta-subunit shares homology with IkappaB and Drosophila Notch protein. GABP binding specificity to mutated N-box in vitro strictly parallels the sequence requirement for beta-galactosidase targeting to the endplate in vivo. In situ hybridization studies reveal that the mRNAs of both GABP subunits are abundant in mouse diaphragm, with preferential expression of the alpha-subunit at motor endplates. In addition, heregulin increases GABPalpha protein levels and regulates phosphorylation of both subunits in cultured chick myotubes. Finally, dominant-negative mutants of either GABPalpha or GABPbeta block heregulin-elicited transcriptional activation of the AChR delta and epsilon genes. These findings establish the expected connection with a presynaptic trophic factor whose release contributes to the accumulation of AChR subunit mRNAs at the motor endplate.

L7 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1999:13173 BIOSIS
DN PREV199900013173
TI ***Promoter*** analysis of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha4 gene: Methylation and expression of the transgene.
AU Watanabe, Hajime; Zoli, Michele; ***Changeux, Jean-Pierre (1)***
CS (1) Neurobiol. Mol., CNRS URA 1284, Inst. Pasteur, 25-28 rue du Dr. Roux, 75724 Paris Cedex 15 France
SO European Journal of Neuroscience, (July, 1998) Vol. 10, No. 7, pp. 2244-2253. ISSN: 0953-816X.
DT Article
LA English
AB Neuronal ***nicotinic*** ***acetylcholine*** ***receptor***

(nAChR) subunit genes compose a family of genes. The major isoform of nAChR in the brain is made up of the alpha4 and beta2 subunits and possesses a high affinity for nicotine. To investigate the mechanisms of the regulation of the nAChR alpha4 gene expression in mouse, its genomic DNA was cloned and characterized. The transcription initiation site was mapped by primer extension and RNase protection experiments and localized at about 254 bp upstream of the translation initiation site. The 5' flanking region of this gene did not have typical TATA box but GC-rich sequences were found around the initiation site. Methylation analysis of this region revealed that genomic DNAs from liver and muscle are partially methylated, whereas little methylation was observed in genomic DNA from brain. To characterize the cis-acting elements driving cell-specific expression of the alpha4 subunit gene, we produced lines of transgenic mice which carry a series of fragments of the alpha4 gene fused with bacterial lacZ as a reporter gene. An 11.5-kb DNA fragment containing 9 kb of the region upstream of the transcription initiation site and the first intron was found to confer an expression pattern which coincides rather well with the endogenous gene expression pattern at early embryonic stages, suggesting that the elements necessary for the onset of alpha4 gene expression are located in this region. A DNA fragment containing the 1.8-kb upstream sequence and the first intron drove expression of lacZ in a limited subset of alpha4 expressing cells, whereas the 1.8-kb upstream sequence alone did not elicit any significant expression. These results show that both upstream and intronic sequences are important for cell-specific expression of the nAChR alpha4 gene.

L7 ANSWER 5 OF 11 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 13786614 INPADOC
TI ***PROMOTER*** OF BETA2-SUBUNIT OF NEURONAL ***NICOTINIC*** ***ACETYLCHOLINE*** ***RECEPTOR***, TRANSGENIC ANIMALS
IN CHANGEUX, JEAN-PIERRE; PICCIOTTO, MARINA; BESSIS, ALAIN
INS ***CHANGEUX JEAN-PIERRE*** ; ***PICCIOTTO MARINA*** ; ***BESSIS*** *** ALAIN***
PA INSTITUT PASTEUR
PAS PASTEUR INSTITUT
DT Patent
PIT NZA COMP. SPECIFICATION ACCEPTED
PI NZ 280674 A 19970922
AI NZ 1995-280674 A 19951214
PRAI US 1994-358627 A 19941214

L7 ANSWER 6 OF 11 MEDLINE DUPLICATE 4
AN 97303230 MEDLINE
DN 97303230
TI The neuron-restrictive silencer element: a dual enhancer/silencer crucial for patterned expression of a nicotinic receptor gene in the brain.
AU ***Bessis A*** ; Champtiaux N; Chatelin L; Changeux J P
CS Neurobiologie Moleculaire, UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur 25/28 rue du Dr Roux, 75724 Paris Cedex 15, France.. abisses@pasteur.fr
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1997 May 27) 94 (11) 5906-11. Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199708
EW 19970803
AB The neuron-restrictive silencer element (NRSE) has been identified in several neuronal genes and confers neuron specificity by silencing transcription in nonneuronal cells. NRSE is present in the ***promoter*** of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** beta2-subunit gene that determines its neuron-specific expression in the nervous system. Using transgenic mice, we show that NRSE may either silence or enhance transcription depending on the cellular context within the nervous system. In vitro in neuronal cells, NRSE activates transcription of synthetic promoters when located downstream in the 5' untranslated region, or at less than 50 bp upstream from the TATA box, but switches to a silencer when located further upstream. In contrast, in nonneuronal cells NRSE always functions as a silencer. Antisense RNA inhibition shows that the NRSE-binding protein REST contributes to the activation of transcription in neuronal cells.

L7 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2000 ACS

AN 1996:422699 CAPLUS
DN 125:78571
TI Genomic DNA fragments coding for the .beta.2-subunit of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** and its transfection into transgenic animals
IN ***Changeux, Jean-Pierre*** ; ***Picciotto, Marina*** ; ***Bessis,*** *** Alain***
PA Institut Pasteur, Fr.
SO Eur. Pat. Appl., 36 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 717105	A2	19960619	EP 1995-402822	19951214
EP 717105	A3	19990407		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CA 2165098	AA	19960615	CA 1995-2165098	19951213
AU 9540373	A1	19960620	AU 1995-40373	19951213
JP 08242866	A2	19960924	JP 1995-326094	19951214

PRAI US 1994-358627 19941214
AB Several genes encoding subunits of the neuronal nicotinic acetylcholine receptors have been cloned and regulatory elements involved in the transcription of the .alpha.2 and .alpha.7-subunit genes have been described. Yet, the detailed mechanisms governing the neuron-specific transcription and the spatio-temporal expression pattern of these genes remain largely uninvestigated. The .beta.2-subunit is the most widely expressed neuronal nicotinic receptors subunit in the nervous system. The structural and regulatory properties of the 5' sequence of this gene were studied. A 1163-bp fragment of upstream sequence is sufficient to drive the cell-specific transcription of a reporter gene in both transient transfection assays and in transgenic mice. Deletion anal. and site-directed mutagenesis of this ***promoter*** reveal 2 neg. and 1 pos. element. The pos. acting sequence includes one functional E-box.
One of the repressor elements is located in the transcribed region and is the NRSE/RE1 sequence already described in promoters of neuronal genes.
The regulatory elements from the .beta.2-subunit-encoding sequences can be used to direct the neuron-specific expression of a nucleotide sequences

encoding a reporter, tumorigenic, oncogenic, or immortalizing foreign
proteins.

L7 ANSWER 8 OF 11 MEDLINE DUPLICATE 5
AN 96313792 MEDLINE
DN 96313792
TI Differential regulation of neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** subunit gene promoters by Brn-3
POU family transcription factors.
AU Milton N G; ***Bessis A*** ; Changeux J P; Latchman D S
CS Department of Molecular Pathology, University College London Medical
School, UK.
SO BIOCHEMICAL JOURNAL, (1996 Jul 15) 317 (Pt 2) 419-23.
Journal code: 9YO. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199611
AB The regulatory region of the neuronal nicotinic acetylcholine (nACh)
receptor alpha 2 subunit gene is activated by the Brn-3b POU family
transcription factor but not by the closely related factors Brn-3a and
Brn-3c. This pattern of regulation has not previously been observed for
other neuronally expressed genes, several of which, such as those encoding
alpha-internexin or SNAP-25, are activated by Brn-3a and Brn-3c but
repressed by Brn-3b. The alpha 3 nACh receptor subunit gene is also shown
to be activated by Brn-3a but is repressed by Brn-3b and Brn-3c. In
contrast, the Brn-3 POU family transcription factors have no effects on
either the alpha 7 or beta 4 nACh receptor subunit genes. The actions of
Brn-3b on the alpha 2 subunit are thus in contrast to the inhibitory
actions of Brn-3b on several promoters that are activated by Brn-3 alpha.
The different actions of the Brn-3 POU factors on the range of nACh
receptor genes tested suggests that the novel stimulation of the alpha 2
subunit by Brn-3b is specific to this subunit and not a general feature of
nACh receptor genes.

L7 ANSWER 9 OF 11 MEDLINE DUPLICATE 6
AN 95318076 MEDLINE
DN 95318076
TI The neuronal ***nicotinic*** ***acetylcholine*** ***receptor***
alpha 2 subunit gene ***promoter*** is activated by the Brn-3b POU
family transcription factor and not by Brn-3a or Brn-3c.
AU Milton N G; ***Bessis A*** ; Changeux J P; Latchman D S
CS Department of Molecular Pathology, University College London Medical
School, UK..
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Jun 23) 270 (25) 15143-7.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199510
AB The regulatory region of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha 2 subunit gene, which
contains six copies of the octamer-related sequence CCCCATGCAAT, is
activated by the Brn-3b POU family transcription factor but not by the
closely related factors Brn-3a and Brn-3c. This effect is in contrast to
the previously documented inhibitory effect of Brn-3b on
octamer-containing promoters that are activated by Brn-3a and Brn-3c.
Activation of the alpha 2 gene by Brn-3b requires that both the POU domain
and other N-terminal sequences are derived from Brn-3b and is dependent on
the intactness of the alpha 2 gene regulatory region, being lost in
truncated derivatives containing one, two, or four copies of the
octamer-related sequence. Surprisingly, however, these truncated
derivatives are activated by Brn-3c. These effects are discussed in terms
of both the influence of the target sequence and its context in the
promoter on activation by the various forms of Brn-3 as well as of
the processes that restrict expression of the alpha 2 subunit gene to a
few cells in the nervous system.

L7 ANSWER 10 OF 11 MEDLINE DUPLICATE 7
AN 96164254 MEDLINE
DN 96164254
TI ***Promoter*** elements conferring neuron-specific expression of the
beta 2-subunit of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** studied in vitro and in transgenic mice.
AU ***Bessis A*** ; Salmon A M; Zoli M; Le Nov`ere N; Picciotto M;
Changeux J P
CS UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur 25/28,
Paris, France.
SO NEUROSCIENCE, (1995 Dec) 69 (3) 807-19.
Journal code: NZR. ISSN: 0306-4522.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X82655; GENBANK-M31433; GENBANK-M90489; GENBANK-M55301;
GENBANK-X63509; GENBANK-L11891; GENBANK-X17102
EM 199606
AB Several genes encoding subunits of the neuronal nicotinic acetylcholine
receptors have been cloned and regulatory elements involved in the
transcription of the alpha 2 and alpha 7-subunit genes have been
described. Yet, the detailed mechanisms governing the neuron-specific
transcription and the spatio-temporal expression pattern of these genes
remain largely uninvestigated. The beta 2-subunit is the most widely
expressed neuronal nicotinic receptor subunit in the nervous system. We
have studied the structural and regulatory properties of the 5' sequence
of this gene. A fragment of 1163 bp of upstream sequence is sufficient to
drive the cell-specific transcription of a reporter gene in both transient
transfection assays and in transgenic mice. Deletion analysis and
site-directed mutagenesis of this ***promoter*** reveal two negative
elements and one positive element. The positively-acting sequence includes
one functional E-box. One of the repressor elements is located in the
transcribed region and is the NRSE/RE1 sequence already described in
promoters of neuronal genes. In this paper, we describe the
neuron-specific ***promoter*** of the gene encoding the neuronal
nicotinic ***acetylcholine*** ***receptor*** beta 2-subunit.

L7 ANSWER 11 OF 11 MEDLINE DUPLICATE 8
AN 93275748 MEDLINE
DN 93275748
TI Negative regulatory elements upstream of a novel exon of the neuronal
nicotinic ***acetylcholine*** ***receptor*** alpha 2
subunit gene.
AU ***Bessis A*** ; Savatier N; Devillers-Thiery A; Bejanin S; Changeux J
P
CS UA CNRS D1284, Department des Biotechnologies, Institut Pasteur, Paris,

France.
SO NUCLEIC ACIDS RESEARCH, (1993 May 11) 21 (9) 2185-92.

Journal code: O8L. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-X69511
EM 199309
AB The expression of the ***nicotinic*** ***acetylcholine*** ***receptor*** alpha 2 subunit gene is highly restricted to the Spiriform lateralis nucleus of the Chick diencephalon. As a first step toward understanding the molecular mechanism underlying this regulation, we have investigated the structural and regulatory properties of the 5' sequence of this gene. A strategy based on the ligation of an oligonucleotide to the first strand of the cDNA (SLIC) followed by PCR amplification was used. A new exon was found approximately 3kb upstream from the first coding exon, and multiple transcription start sites of the gene were mapped. Analysis of the flanking region shows many consensus sequences for the binding of nuclear proteins, suggesting that the 1 kb flanking region contains at least a portion of the ***promoter*** of the gene. We have analysed the negative regulatory elements present within this region and found that a silencer region located between nucleotide -144 and +76 is active in fibroblasts as well as in neurons. This silencer is composed of six tandem repeat Oct-like motifs (CCCCATGCAAT), but does not bind any member of the Oct family. Moreover these motifs were found to act as a silencer only when they were tandemly repeated. When two, four or five motifs were deleted, the silencer activity of the motifs unexpectedly became an enhancer activity in all cells we have tested.

=> d his

(FILE 'HOME' ENTERED AT 13:32:54 ON 22 SEP 2000)

FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 13:33:44 ON 22 SEP 2000
E CHANGEUX JEAN-PIERRE/AU
L1 408 S E1-E2
E PICCIOTTO MARINA/AU
L2 72 S E2-E5
E BASSIS ALAIN/AU
E BESSIS ALAIN/AU
L3 81 S E1-E3
L4 514 S L1 OR L2 OR L3
L5 136 S L4 AND NICOTINIC ACETYLCHOLINE RECEPTOR/AB,BI
L6 27 S L5 AND PROMOTER/AB,BI
L7 11 DUP REM L6 (16 DUPLICATES REMOVED)

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		38.52	38.82

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		-0.56	-0.56

STN INTERNATIONAL LOGOFF AT 13:37:14 ON 22 SEP 2000